# EMPHYSEMA OF THE LUNGS*

BY

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PART II

### Physical Signs

The mechanism by which loss of pulmonary elasticity may lead to enlargement of the thoracic cage has already been discussed. The diaphragm, because it is no longer pulled upwards by the lungs, descends and becomes less convex, and its movement on inspiration is greatly diminished. Because of its loss of tone the diaphragm may also present an irregular outline in the radiograph. The intercostal muscles will also be unable to perform their proper function, as they are concerned with moderate inspiration and the chest is already expanded when inspiration begins: to expand the chest further the accessory muscles will have to be used, particularly the pectorals, and these raise the front of the chest as a whole in a "heaving" manner, such as occurs in a normal individual towards the end of a forced inspiration. Thus inspiration has to be performed by means of an unnatural inspiratory effort, and the patient with emphysema will encounter even greater difficulty in deflating his lung. Normally, expiration is largely, if not wholly, a passive act: the thoracic cage is pulled inwards by the elastic recoil of the lung. With loss of elasticity there must be loss of elastic recoil, so that if the lung is to be deflated it has to be squeezed. The respiratory musculature was not built for this task, and the intercostals have to be assisted by the accessory muscles of expiration: the muscles of the abdominal wall can often be felt to contract on expiration, which is prolonged as it is in other conditions, such as asthma and tracheal obstruction, in which the lungs have to be compressed by an active muscular effort. With so extensive an impairment of both inspiration and expiration it is not surprising that the vital capacity and chest expansion are reduced.

Many of the physical signs which can be elicited from the lung itself can be explained on the basis of loss of elasticity. I have already emphasized that, in the inelastic lung, distribution of expansion might be expected to be far from equal and should be greatest where the force is applied, which is on the peripheral parts under the visceral pleura. It would be reasonable to expect that, as the application of this force is repeated many thousands of times daily, the superficial air sacs next the pleura would in time become over-distended. With over-distension their supporting framework would be lost and further stretching would become easier, so that a true vicious circle might be established. It is easy to imagine that this mechanism could be the cause of the over-distension of superficial air sacs, with bulla formation, which occurs in emphysema, although the usual explanation given is that cough distends these air sacs. It is obvious, however, from the mechanism of coughing, which is discussed below, that the lung is compressed rather than distended, and it does not seem possible that cough could be responsible for the formation of bullae, except perhaps in poorly supported areas such as the apex of the lung. Presumably it is this superficial over-distension of air sacs and bulla formation which causes the remarkable hyperresonance of the percussion note—a note which is often more resonant than over a pneumothorax. Why this should occur has not been explained, but it may well be that the over-distended air sac acts as a sound-box or resonating chamber, in the same way as does a cavity. It is also easy to imagine how this distension might occur, more particularly along the edges of the lung, where so-called pleural sinuses exist. With each inspiratory effort on the part of the pectoral muscles the anterior chest wall is pulled outwards and upwards as a whole, and much of this force must be expended on pulling the lung into the pleural

sinuses so as to fill the potential gap between the chest wall and the heart. In time this part of the lung will become over-distended, so that there is a gap between the heart and the chest wall, filled by lung. This space can be clearly seen in the lateral view of a radiograph, and it explains a variety of physical signs. The absence of the apical impulse, loss of cardiac dullness, and distant heart sounds necessarily follow. The expansion of the lung, so that it covers the heart in considerable depth, may also in part account for the increase in antero-posterior diameter and the barrel shape of the chest. Similarly the loss of liver dullness must in part be caused by expansion of the lung into the pleural sinuses, although the low position of the diaphragm is of course another factor.

All these changes can be explained directly or indirectly on the basis of loss of elasticity ; but it should not be forgotten that enlargement and deformity of the thorax and its contents are partly dependent on other factors, such as the elasticity and mobility of the thoracic cage. These factors vary with the age and build of the patient, and are presumably responsible for the inconstancy and unreliability of the physical signs of emphysema. There remain several physical signs which have not been discussed or explained. Breath sounds are usually faint, especially at the bases, where they may be inaudible ; but as I do not know how vesicular breath sounds are produced in health it is difficult to discuss the reason for their suppression. A possible explanation, held by some Continental writers, is that vesicular breathing is produced by the jet of air thrown into the atrium from the alveolar duct. The absence of this mechanism in emphysema has already been discussed, and may be responsible for the suppression of breath sounds. Vocal resonance and fremitus are sometimes decreased in emphysema. I have no explanation to offer for this, as I do not understand the mechanism of conduction of the spoken voice in the healthy lung, although I know of several theories which do not fit the facts.

There are several factors which may contribute to the formation of the barrel-shaped chest, and, although these are not entirely independent of one another, their relative importance probably varies from patient to patient. I have already described how loss of elasticity contributes two factors towards this change: the first was the inspiratory position of the chest, and the second the expansion of the lung between the heart and the sternum. A third important factor must be the dorsal kyphosis, which is distributed throughout the thoracic vertebrae, and which may produce an increase in the antero-posterior diameter by the same mechanism as in Pott's disease. There are thus at least three separate causes for the deformity of the chest, and another factor must be the diminishing flexibility of the chest wall with advancing age. It is not surprising, therefore, that the barrel-chest phenomenon is variable, being marked in some cases and inconspicuous in others.

Thus loss of elasticity alone could directly or indirectly cause the dyspnoea of emphysema, almost all the physical signs, and a considerable proportion of the morbid anatomical appearances. There are, however, a number of changes characteristic of emphysema which I am unable to explain on the basis of loss of elasticity. These are the destruction and degeneration of alveolar walls and blood vessels, the degeneration and atrophy of the pleura, and the degeneration with loss of elasticity of the costal cartilages and the thoracic vertebral cartilages. All these structures are part of the respiratory apparatus, and it is reasonable to suppose that the cause of this widespread damage is also the cause of the degeneration of the elastic fibres themselves. The nature of this destructive agent is discussed below.

### Aetiology

In seeking the fundamental cause of emphysema I have described the fallacy of thinking in terms of over-distension and overstretching of the lungs. The lungs as a whole are not overstretched, and we should think rather of some agent which produces destruction and degeneration of many of the structures concerned in respiration, both in the lung and outside it. With this in mind I propose to review briefly the various theories that have been suggested as the cause of emphysema.

The expiratory theory is that the air sacs become overdistended because they cannot be emptied. The inspiratory

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theory claims that over-distension is produced because too much air is drawn into the lungs by the increased inspiratory effort which precedes coughing and which may be associated with asthma, manual labour, and occupations such as glass-blowing and the blowing of wind instruments. There is also a theory that emphysema is primarily due to a faulty position of the chest and that it is the enlargement of the thoracic cage which causes overstretching of the lung. Many criticisms have been levelled against these three theories, but I will only repeat that, from the evidence already described, all of them appear to be based on the false assumption that the lung as a whole in emphysema is truly overstretched and over-distended.

It has been claimed, from quite unconvincing evidence, that local impairment of nutrition is the cause of emphysema. I have pointed out that under resting conditions there is no diminution in the total amount of blood flowing through the lungs, and it is also true that in other diseases there may be more extensive atherosclerosis than is seen in emphysema, although the lung does not become emphysematous. For these reasons it is improbable that the vascular changes in the lung are of any aetiological significance.

It has been suggested that the lung changes are primarily degenerative or atrophic; but this is too easy a way of evading the issue, as it by no means explains the many aetiological factors known to be involved. The most obvious of these concerns respiratory obstruction. Almost all cases of emphysema have a long-standing history of chronic bronchitis or asthma, or both, and this is also true of emphysema in horses and cattle. The effects of cough and bronchial spasm on the lung, and their mechanism, are therefore of primary importance in any discussion on the aetiology of this disease. The mechanism of coughing is simple. There is a short inspiratory effort which by no means fills the chest to full capacity; the glottis is then closed, and the lungs are forcibly compressed by all the muscles of expiration, so that the pressure of air in the alveoli may rise to over 50 mm. of Hg (Kountz and Alexander, 1934; Rasmussen and Adams, 1942). The glottis is then opened and the strain on the ribs, the pressure on the pleura, and the pressure in the alveoli are suddenly released. In the case of the alveoli there must be a wave of pressure change passing from the bronchi out to the periphery of the lung. It is presumably this sudden release of pressure which is responsible for the sensation, usually experienced, that the lung is being distended. It is obvious, however, from the mechanism of coughing, that the lung is compressed and not in any sense distended. This building up of pressure followed by its sudden release must mean increased stress and strain on all the structures involved, especially on the alveolar walls, which are not robust and through which passes this wave of pressure change. A chronic bronchitic may cough scores of times in a day, and it seems reasonable to suppose that this type of stress and strain, or wear and tear, repeated over many years, could in time produce the loss of elasticity and degenerative changes observed in the alveolar walls. In many respects there is an analogy in hypertension, in which, without any overstretching of the blood vessels, there may be loss of elasticity and other degenerative changes, and these are ascribed by recent investigators to the chronic stress of raised pressure. Outside the lungs it is also reasonable to suppose that wear and tear might occur when cough is chronic. The pleura might suffer for the same reason as the alveoli, and the thoracic cage is also strained: one has only to place a hand over the costal cartilages while coughing to realize that the impact is considerable.

In asthma there may be the same degree of change in intra-alveolar pressure as in coughing, and although the change is not so sudden, it is repeated more often (v. Neergaard and Wirz, 1927; Hartwich, 1930). This stress and strain repeated many thousands of times a day should have the same effect as chronic cough. The mechanism of the experimental production of so-called emphysema by tracheal stenosis in animals is presumably the same (Paine, 1940a, 1940b). It is also true that during an acute attack of bronchial asthma the increased expiratory resistance may lead to distension of the lung, which tends to subside when the attack is over. This temporary distension does not rupture the alveolar walls, or in fact lead to any permanent damage, but it probably does add to the stress and strain produced by the other factors I have described.

Almost all patients with emphysema suffer from either chronic bronchitis or asthma, and in these cases the explanation of the pathogenesis of emphysema which I have given is the most likely one. It is also true that some patients with chronic bronchitis or asthma develop emphysema comparatively rapidly, while others never become emphysematous. The forcefulness of the cough is obviously an important factor, and age is another. To discuss the changes in resistance to wear and tear or to trauma that occur in senescence would in itself occupy several lectures, but no one would dispute that with age there is a decrease in the resistance of the tissues. This is particularly true of the elasticity of the blood vessels; and with advancing years there may also be diminished resistance in the lungs, and in the elastic structures of the thoracic cage, to the stress and strain of cough or of asthma, and this diminution in resistance may take place earlier in some individuals than in others.

There still remains to be explained the unusual case of emphysema with no previous history of asthma or chronic bronchitis. I have seen three such cases when shortness of breath preceded the onset of cough, and in one the diagnosis was confirmed at necropsy. I can only suggest that, very rarely, the stress and strain on the lung which occurs with ordinary breathing may be sufficient to destroy its elasticity.

Other aetiological factors have been suggested, and a hereditary tendency to emphysema is one. This may be so, but I have not seen it except indirectly as a possible familial tendency to asthma and chronic bronchitis. Hard manual labour is another, but my own belief is that this is only of importance in that it increases the incidence of chronic bronchitis and accelerates the process of ageing. It is also a striking testimony to the conservatism of medical teaching that there is scarcely a textbook of medicine which does not quote glass-blowing and the blowing of wind instruments as probable or possible causes of emphysema; and this in spite of ample evidence in the literature that no such aetiological relationship exists (Christie, 1939). The original statement of Laennec, made in 1819, based on no actual cases, has been copied from textbook to textbook over a period of 120 years.

### Diagnosis

I believe it is those with little clinical experience who think they have a clear conception of the diagnosis of emphysema. The pathology of the disease as taught is quite precise and the description of its clinical manifestations is equally so, but the differential diagnosis is not even mentioned in most textbooks. It is those with wide clinical experience who have expressed difficulty in interpreting the so-called physical signs of emphysema. I have discussed the diagnosis of emphysema with many clinicians, and have found a wide divergence of opinion as to the reliable signs of this disease. All were agreed that the physical signs as traditionally described could be of value in diagnosis but could also be misleading. Obliteration of the cardiac dullness, diminished chest expansion, reduction in vital capacity, and the radiograph were each suggested as being most valuable in doubtful cases. I can find no agreement—in fact, there is wide disagreement—with regard to the relationship of the barrel-chest phenomenon to emphysema. Some believe, and teach, that the physical signs associated with the barrel-shaped chest, if definite, necessarily mean emphysema, although they admit that the lesion may neither progress nor lead to symptoms. Others believe that the barrel-chest phenomenon may occur quite independently of emphysema, and that in these circumstances it is of no significance. Such a divergence of opinion as to the diagnostic criteria naturally makes for scepticism, and attempts to correlate the clinical diagnosis of emphysema with the facts as revealed by necropsy have led to further doubts. It has been stated that only a small proportion of those diagnosed at necropsy are recognized during life, and it has also been shown that many of those with barrel-shaped chests furnish no evidence of emphysema when they come to the post-mortem table (Cabot, 1927; Davidson, 1936; Roelsen, 1938). For this reason some writers have gone so far as to say that the physical signs of emphysema are meaningless. I believe that this is certainly untrue, just as I believe that the textbooks are misleading, to say the least, in the neat array of physical signs which they present.

In order to determine the reliability of these physical signs Dr. P. Hill and I have reviewed 72 cases diagnosed on the post-mortem table as generalized hypertrophic emphysema. All of these cases came from the medical wards of the London Hospital, but an accurate and detailed case history was not always available, as some were moribund on admission. A history of chronic cough was given in 94%, and in a quarter of these it was associated with mild or severe asthma. Dyspnoea on exertion was present in 78% of the 45 case histories in which mention was made of the presence or absence of this symptom. In the majority of cases, however, the physical signs of emphysema were not observed. In 35% there was enough evidence to suggest an " emphysematous chest," but in only 13% could this evidence be said to be complete. This series of cases suggests that a more reliable guide to a diagnosis of emphysema than the physical signs is a history of chronic cough or asthma, associated with dyspnoea on exertion. I have therefore investigated another series of 25 patients who gave such a history and in whom no cause of dyspnoea other than emphysema could be discovered. In 92% of them two or more of the physical signs of emphysema were present, and in these a diagnosis of " emphysematous chest " might have been made ; in 90% expiration was prolonged, but the incidence of any other individual physical sign was not more than 72%, and only 44% showed the traditional picture of emphysema. The average age of these patients was 54 years, and as a control to this series I have analysed a group of 40 patients, all over the age of 45, none of whom had a history of either cough or dyspnoea. In 9 of them the chest might have been said to be " emphysematous," and 4 presented the classical physical signs of emphysema.

The only conclusion that can be drawn from these series of cases is that the signs of emphysema are unreliable ; they may be absent in patients suffering from the disease, and are not uncommonly present in patients without emphysema. Several special diagnostic methods have been recommended ; but these are seldom used, and their reliability has not been established. The vital capacity in emphysema is usually diminished. I have made this measurement on 25 cases, all of them with a history of chronic bronchitis and dyspnoea on exertion. In 20 the vital capacity was less than 70% of the normal standard, as calculated from the surface area, and in 13 it was less than 60%. There is a natural tendency during senescence for the vital capacity to diminish ; but at any age a vital capacity below 60% of the normal is significant, as is less than 75% of normal before the age of 50.

The diagnostic value of the vital capacity is enhanced if this measurement is made on the type of recording spirometer which is employed in the routine estimation of the basal metabolic rate. Such a record not only gives a measure of the vital capacity but can also be used to indicate the loss of elastic recoil in the emphysematous lung. I have discussed these tests in detail elsewhere (Christie, 1934 ; Meakins and Christie, 1934) ; they are based on the fact that when a normal individual takes a deep breath his lungs recoil passively to their original size because of their elasticity. In emphysema this recoil is abolished or impaired, and the lung has to be compressed by the muscles of expiration. After a full inspiration, therefore, the lung is seldom deflated to its original size when the patient is asked to expire forcibly. There are two other manifestations of loss of elasticity which can be demonstrated by this type of respiratory tracing: after a full inspiration the patient is incapable of deflating his lung as efficiently as after an inspiration of moderate depth, and the level at which the patient breathes is irregular.

During the past ten years I have performed this test on 66 patients with chronic bronchitis and dyspnoea on exertion: in 30 these characteristics were obvious and constant, in 26 the tracings were suggestive of loss of elastic recoil, and in the remaining 10 no abnormality could be detected. Of 116 controls, 11 would not co-operate, none showed definite loss of elasticity, and only 4 were suggestive of slight loss of elasticity. The main value of these respiratory tracings is that it is only patients with emphysema who consistently show loss of elastic recoil ; admittedly this is true of only about 50% of cases, but most of the others give tracings which are suggestive. The drawbacks of this test are obvious. Except in hospitals where

the basal metabolic rate is measured by means of a recording spirometer, special equipment is required. The test has to be performed by someone with special knowledge of respiratory tracings, patience is required to ensure that the subject knows what is wanted of him, and I do not expect that this test will be used except perhaps by those who have a particular interest in emphysema.

It is easier to measure the chest expansion than the vital capacity, but unfortunately this also diminishes with age. In my experience about 60% of patients with emphysema have a chest expansion of less than one inch, but I have seen it reduced to half an inch in a man aged 65 in whom there was no other evidence to suggest pulmonary or cardiovascular disease.

In recent years the radiograph has been suggested as a reliable guide to emphysema. This has not been my experience. In 11 cases in which the diagnosis was confirmed at necropsy, radiographs suggested emphysema in only 5. In 25 other cases, in which diagnosis was made on clinical grounds, the value of the x-ray film was approximately the same.

A diagnosis of emphysema based only on physical signs is therefore unreliable, since these depend largely on the barrel-chest phenomenon, which may occur independently of this disease. In my opinion the diagnosis should only be considered certain when dyspnoea on exertion, of insidious onset, not due to bronchospasm or left ventricular failure, appears in a patient who has some of the physical signs of emphysema together with chronic bronchitis or asthma. The absence either of dyspnoea or of physical signs should cast doubt on the diagnosis, but should not exclude it ; the more physical signs present in a patient with chronic bronchitis or asthma, the greater is the likelihood that he has emphysema. I must confess, however, that in the absence of dyspnoea I am always uncertain as to diagnosis and prognosis, but when dyspnoea of the type described above appears in a patient with chronic bronchitis I am very seldom in doubt. A large series of cases will have to be followed up over many years before the necessary criteria for early diagnosis can be established. Until this has been done I feel that the terms " emphysematous " and " emphysematous chest " should be discarded, as it is their use which has been partly responsible for the lack of progress in the understanding of emphysema since its classical description over a century ago by Laennec. These terms are quite misleading, and should be replaced by some such expression as " barrel chest " or " barrel-chest phenomenon," reserving the diagnosis of emphysema for those patients who have the symptoms as well as the signs of the disease. I feel that if this were done the chances of progress would be increased, particularly with regard to the use of special diagnostic procedures.

### Treatment

The treatment of emphysema is essentially symptomatic, as elastic tissue cannot regenerate and nothing can restore the structure of the lungs. Until heart failure supervenes, the only symptom, other than cough, is dyspnoea on exertion, and I will first emphasize the value of ephedrine. Although there may be no evidence of bronchospasm or resistance to respiration, the administration of ephedrine not infrequently relieves the dyspnoea of emphysema. A possible explanation of this effect is that the bronchioles leading to the over-distended air sacs and bullae are less capable of changes in calibre than those leading to healthier parts of the lung ; bronchospasm, although not clinically manifest, would in this case increase the proportion of the inspired air deflected to these useless parts of the lung, and the relief of bronchospasm with ephedrine would improve the efficiency of ventilation and thus relieve dyspnoea.

Several procedures, the purpose of which is to deflate the lung, have been described, and it is said that these increase the efficiency of respiration. Pneumothorax is one, and relief of dyspnoea from this procedure, both in man and in horses suffering from this disease, has been claimed. More popular in this country, and less dangerous, are respiratory exercises designed to teach the patient to deflate the lung and to increase the use of the diaphragm. A well-fitting abdominal belt will also raise the diaphragm by increasing intra-abdominal pressure, and, more recently, pneumoperitoneum has been suggested for the same purpose. All these procedures have been recom-

mended, and various theories have been put forward to explain the beneficial effects observed. Two effects are common to most of them. First, the diaphragm is raised so that its convexity is increased and its efficiency therefore enhanced, and it was for this purpose that I first suggested the use of the abdominal belt. Secondly, the lung is deflated by these procedures so that it contains less air. The decrease in volume is, however, very small—so small that it can hardly be measured—and the beneficial effects are probably for the most part due to increased efficiency of the respiratory musculature. It is also possible that with greater collapse of the superficial bullae efficiency of ventilation may be increased.

When heart failure supervenes oxygen should be given, as the added insult of anoxia to a heart that is failing for other reasons greatly lessens the chances of recovery. Recovery from heart failure in emphysema is uncommon, but I have recently had a patient who recovered from two attacks, during both of which he was moribund. In the first oxygen was administered continuously for one week, and in the second for 10 days. He subsequently died during a third attack, and the diagnosis of emphysema was confirmed at necropsy.

The treatment of emphysema is thus far from being hopeless, although it is essentially symptomatic. Ephedrine should be tried in all cases. Respiratory exercises or an abdominal belt, or both, may increase considerably the tolerance to exercise. And lastly, when heart failure supervenes oxygen therapy is of the greatest value.

### Summary
In this paper the disturbance of function in emphysema, and its mechanism as revealed by experiment at the bedside, are emphasized, and the bearing of these clinical observations on the natural history of the disease and its diagnosis is discussed. The primary lesion is loss of elasticity, the most common cause of which is the stress and strain of cough or respiratory obstruction on the structures concerned in respiration, the process being comparable to the loss of elasticity which occurs in blood vessels following hypertension. Almost all the symptoms and signs of emphysema, and also the over-distension of air sacs and formation of bullae, can be explained on the basis of loss of elasticity, and it is suggested that it is the wastage of ventilation on this " pathological dead space " which is responsible for the dyspnoea and the impairment of haemo-respiratory exchange which occur. The difficulties and fallacies in the diagnosis of emphysema are described, and it is suggested that the present state of confusion would be diminished if the diagnosis were reserved for those who have symptoms as well as signs of this disease.

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# INFANT FEEDING IN RELATION TO MORTALITY IN THE CITY OF BELFAST

BY

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In the course of a recent study of the infant mortality problem in Belfast we have collected some facts relating to infant feeding. For this city the death rate of children under 1 year has been high, and during the past four years has ranged from 85 to 122 per thousand live births. In the survey data were obtained concerning 554 deaths, or 84% of the total deaths of legitimate infants occurring from June, 1941, to June, 1942, in Belfast (completed by Dec., 1942). The total legitimate births in Belfast for this period were 7,778, 3,359 occurring in the first six months. Material was also collected from a group of children who had survived 1 year of life in the city. This control sample was established by selecting every fifth child born during the first six months of the period, and, allowing for wastage, finally numbered 477.

In order to maintain constant values the information was obtained by one investigator (E. T. M.) from a personal visit to the parent or guardian. Parents usually possess vivid memories of the circumstances of the death of their children, and were generally willing to discuss the matter. Cases were excluded when the fatal illness was due to hardships resulting from previous air raids, and when families had moved from the city and could not be followed up. Information was recorded concerning the size of the family (and previous infant deaths), income, housing, the care and feeding of the child, domestic hygiene, the ante-natal and obstetric attention, cause of death, the medical, nursing, hospital, and other factors relating to the fatal illness—entailing more than 40 questions in each case. In the control group somewhat similar data were obtained. This paper deals only with the information we have secured concerning infant feeding.

### Infant Feeding in Belfast
Feeding is an important factor associated with infant mortality, and in any examination of the problem a knowledge of the general character of the infant feeding practised is necessary. This subject was considered under three main headings—viz.: (1) the principal foods given to ordinary children for the first six months of life; (2) the food given for the second six months; and (3) foods the infants received before the fatal illness. It is obvious that this method of inquiry does not yield results in great detail, but it will present a fairly accurate picture of the main methods of infant feeding used in the city.

TABLE I.—*Analysis of Feeding of Infants in the Control Group, during the First Year of Life*

| Method of Feeding | Aged 0–6 months | Aged 6–12 months |
|---|---|---|
| Breast-fed (with or without " supplement ") | 337 (71%) | 157 (33%) |
| Cow's milk | 61 (13%) | 33 (7%) |
| Dried proprietary milk | 40 (8%) | 19 (4%) |
| Cereal foods | 39 (8%) | 58 (12%) |
| Cereal and " mixed feeding " | — | 68 (14%) |
| " Mixed feeding " and cow's milk | — | 142 (30%) |
| Total | 477 (100%) | 477 (100%) |

For the children in the control group, which is representative of living children in Belfast, we found (see Table I):

1. *During the first six months* almost three-quarters received breast milk, with or without a " supplement," as the principal food. Of the remainder, a slightly larger proportion were fed on cow's milk than on cereals or dried proprietary milk.

2. *During the second six months* the same children showed a change in diet, with a decrease in breast-feeding. Only one-